VOL. 32 JULY, 1905 No. 7

THE

Journal

OF

Nervous and Mental Disease

Original Articles

PRESIDENTIAL ADDRESS.

THE IMPORTANCE IN CLINICAL DIAGNOSIS OF PARALYSIS OF ASSOCIATED MOVEMENTS OF THE EYEBALLS (BLICKLAEHMUNG), ESPECIALLY OF UPWARD AND DOWNWARD ASSOCIATED MOVEMENTS.[1]

BY WILLIAM G. SPILLER, M.D.,

PROFESSOR OF NEUROPATHOLOGY AND ASSOCIATE PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

To preside over this distinguished body of American neurologists is an honor that your chairman feels too deeply to express adequately in words.

"* * * like a cipher,
Yet standing in rich place, I multiply
With one 'We thank you' many thousands more,
That go before it."

Our membership list now is almost full, and we shall be obliged to raise the number above one hundred or to reject desirable applicants. There is some danger in doing the former. When there are many vacancies a candidate is more likely to be accepted without careful scrutiny of his record than when admission is of necessity limited. On the other hand it would be unfortunate to bar out desirable men. A moderate increase in our membership may be advantageous, and the Council will suggest the placing of the limit at 110.

---

[1] Read at the meeting of the American Neurological Association, June 1, 1905. From the Neuropathological Laboratory of the University of Pennsylvania.

It is proposed to make some important changes in the Congress of American Physicians and Surgeons. The subject has already come before this Association but must be considered again. The following resolution is offered: Resolved: That in the opinion of this Society it is desirable that the Executive Committee of the Congress of American Physicians and Surgeons of 1907 should consider the advisability of altering the constitution of the Congress, and report progress thereon to the several Societies interested at their sessions of 1905 and 1906.

In preparing this address it has seemed best to me to follow a custom that on occasions such as this has been more common abroad than at home, and to discuss a subject of general interest. I therefore present for your consideration the results of some investigations that may in a small degree increase our knowledge of the disorders of the nervous system.

Disturbances in the associated movements of the eyes (Blicklähmung) afford a sign of localizing value in lesions of the brain, and yet this subject has received comparatively little attention. V. Kornilow, who has written the best recent paper on these palsies, remarks that clinical cases carefully studied are rare, and even more so are those with necropsy.

The opinion of Parinaud and Sauvineau that upward or downward associated ocular paralysis is always associated with paralysis of convergence is incorrect, as shown by a case reported by Parinaud himself, by a case reported by v. Kornilow, and by other cases. It is true, however, as Parinaud pointed out in 1883, that in paralysis of associated ocular movements diplopia often is absent. Indeed when in such palsy diplopia later appears it may be a sign of improvement, as it was in one of the cases of my series (Case 5), where the disappearance of the palsy in one eye caused diplopia, soon followed by complete recovery. Although paralysis of associated ocular movement of any form is usually a sign of grave significance, one should be careful about giving a fatal prognosis. In a case reported by Raymond and Cestan[2] the paralysis lasted ten years, and in a number of cases complete recovery has occurred (Case 5 of my series, and other cases), while in others improvement has been very marked (Case 8 and Case 9, of my series).

---

[2] Raymond and Cestan. Revue Neurologique, 1901, p. 70.

Having had the opportunity recently, through the kindness of many colleagues, to whom credit is given in the detailed reports, to study four cases (Beier,[2] Cases 1, 2 and 3), in which paralysis of lateral associated ocular movements occurred, and nine cases in which paralysis of upward or downward associated ocular movements was a striking sign (including three of the four cases mentioned above); having also examined microscopically the material obtained from four of these cases, I have selected paralysis of associated ocular movements as the subject of my address. I shall not devote much space to the study of the paralyses of lateral associated movements, as these have received much attention, and I[3] have recently discussed this subject. The evidence is strong that this form of paralysis is indicative of a lesion of the posterior longitudinal bundle near the sixth nucleus. The external rectus muscle on the side of the lesion may be more affected than the internal rectus of the other eye, and this according to Parinaud[4] is the rule. He mentions also that the movement of convergence may be greater in the eye in which the external rectus is paralyzed, because of the paralysis of the external rectus.

The disturbances of the upward or downward associated movements have been studied less than those of lateral associated movement. The former, though usually acquired, may be congenital, and Oppenheim[5] has observed congenital paralysis of associated upward movement. Uhthoff[6] speaks of paresis of upward associated movement with slight impairment of movement in other directions, occurring in multiple sclerosis.

We have reason to believe that the cerebral cortex exerts some control over the associated movements of the eyeballs; especially strong is the evidence concerning lateral conjugate movements. Conjugate deviation of the eyes has been studied within the past few years by Klaas,[7] and from his investigations of the literature, it appears that this phenomenon was first observed by Andral (1834). It was studied by Duplay, Durand-Fardel and others, but Prevost showed that conjugate deviation of the eyes often occurs in hemiplegia and has nothing to do with strabismus, as Andral believed; he showed further that it is often associated with deviation of the head; that the deviation is toward the lesion when the lesion is cerebral, and away from the lesion when it is in the isthmus. Landouzy believed that irritation and paralysis produce deviation in opposite directions when the lesion is in the cerebrum, and Grasset showed that the patient looks at his paralyzed limb when the lesion is a paralyzing one in the brain stem, and at the lesion when it is an irritating one, and in this part of the brain. Wernicke assumed that the rapid disappearance of conjugate deviation of the head and eyes is owing to the partial control of the lateral ocular movements by the cerebral hemisphere of the same side, and to the action of this hemisphere in place of the injured opposite cerebral hemisphere. Wernicke believed that conjugate deviation of the eyes is caused by a lesion of the lower parietal lobe, and he reported three cases of lesion in the lower part of each parietal lobe in which voluntary lateral associated ocular movements were lost, pseudo-ophthalmoplegia, although in another case with similar lesions disturbance of the ocular movements was not observed. The evidence concerning a center for lateral movements of the eyes in the lower parietal region or angular gyrus is conflicting. There is more evidence that a center for lateral associated ocular movements is situated in the posterior part of the frontal lobe.

---

[3] Potts and Spiller. Univ. of Penna. Med. Bul., Dec., 1903.

[4] Parinaud. Archives de Neurologie, Vol. 5, No. 14, 1883, p. 145.

[5] Oppenheim. Lehrbuch der Nervenkrankheiten, 4th Ed., Vol. 2, p. 706.

[6] Uhthoff. Archiv. für Psychiatrie, Vol. 21, 1890, p. 379.

[7] W. Klaas. "Ueber konjugierte Augenablenkung bei Gehirnkrankheiten," Inaugural Dissertation, Marburg, 1898.

The evidence concerning a center for lateral movements of the eyes in the lower parietal region or angular gyrus is conflicting. There is more evidence that a center for lateral associated ocular movements is situated in the posterior part of the frontal lobe.

The recent investigations of Grünbaum and Sherrington by electrical irritation have again demonstrated that a center for ocular movements probably exists in the frontal lobe. Pathological alteration of the frontal lobe has given evidence to the same effect. In a case of cerebral syphilis with severe lesion of the right frontal lobe, reported by Karl Schaffer,[8] paralysis of associated movement of the eyeballs to the left occurred, and Schaffer concludes from a study of this case that a center for lateral associated movements exists in the posterior part of the second frontal convolution, although he does not deny that there may be another and similar center in the supramarginal and angular gyri.

---

[8] Schaffer. Neurogisches Centralblatt, Nov. 16, 1904, p. 1035.

Hemianopsia cannot be regarded as the cause of conjugate deviation in all cases, although much has been attributed to it recently by certain French writers. Dejerine and Roussy[9] have demonstrated that it is not necessarily the cause of the deviation, inasmuch as deviation existed in a patient of theirs who had been born blind and whose cortical visual zone had not been developed.

So far the knowledge gained relates chiefly to associated lateral movements, but we know much less concerning a cortical center for upward or downward associated movements.

The views of Parinaud and some other writers on this subject lack confirmation. They believed that there is a center for ocular movements in the occipital lobe and a more important one in the frontal lobe. The inferior part of the latter center controls the upward movement of the eyes, the superior part controls the downward movement of the eyes, the intervening part controls the lateral movements. It is true that lateral associated movements have been impaired with paralysis of upward or downward associated movements in a number of instances, as they were in some of my cases, but a lesion in the vicinity of the corpora quadrigemina will better explain this form of paralysis than a lesion of the cerebral cortex. Teillais says regarding his own case, it would be unreasonable to regard the lesion as cortical, because if the lower part of the cortical center controls the upward movements, and the upper part the downward movements, then the whole of this center except the middle portion must have been destroyed, as the lateral movements were preserved. Such a view cannot be accepted.

Paralysis of lateral associated movement from cortical lesions, as seen in the conjugate deviation of the eyes, is always, so far as I know, transitory, and the persistence of such paralysis points to a lesion of the posterior longitudinal bundle. We have little or no anatomical or pathological evidence that is really valuable concerning the existence of a cortical center for upward or downward associated movements, but in reasoning from analogy we must assume that such a center or centers exist. The clinical case that Parinaud[10] reported in 1892 as indicating a lesion of the frontal center, is in the light of our present knowledge unsatisfactory because of the association of homonymous lateral hemianopsia.

---

[9] Dejerine and Roussy. Revue Neurologique, Feb. 15, 1905, p. 161.

Parinaud reported the case before the Society of Ophthalmology, April 5, 1892. The man had paralysis of the lower left part of the face, of the left half of the tongue, and temporarily of the left hand. He had almost complete paralysis of the elevators of the eyeballs, with a little reduction in the extent of the lateral movements. Convergence was well performed. (Here Parinaud presents a case of paralysis of upward associated movement with preservation of convergence.) Downward movement was normal. Parinaud believed there was a lesion in the right frontal lobe, at the level of the lower part of the Rolandic fissure, involving the centers for the face, tongue, hand, and associated movements of the eyeballs.

This patient had transitory partial left homonymous hemianopsia, and this was supposed to be the result of a lesion near the visual zone, which occupies, according to the teaching of Parinaud in 1892, all the occipital lobe, and extends to near the ascending parietal convolution. This opinion we now can hardly accept.

Hysteria may cause paralysis of the associated ocular movements, but there is no pathognomonic sign of this form of the palsy, and in some instances a correct diagnosis may be extremely difficult or even impossible. No case presents this difficulty more clearly than the one reported by Crouzon, Marie and Babinski[11] before the Neurological Society of Paris on three different occasions in 1900 and 1901.

Crouzon presented the case as one of spasmodic tic, later Babinski and Parinaud expressed the opinion that it was organic and caused by a supranuclear lesion, and still later Marie presented the case with the diagnosis of a purely functional disorder, a neurosis, as a spasm of the elevators of the eyeballs. When the patient threw his head as far backward as possible and followed with his eyes a finger lowered slowly the eyeballs moved downward, but if his head were erect and he attempted on command to look at his feet, he bent his head forward and the eyeballs went forcibly upward. After thirty or forty seconds the spasm disappeared and the eyeballs returned to the normal position. Marie from this concluded that there must be a spasm of the elevators and not a paralysis of the depressors. The disorder had occurred after an apoplectic attack lasting seventeen hours, and the man had become blind. During the attack he had not had stertorous breathing nor passage of urine or feces, and this Marie regarded as remarkable if there were an organic lesion. The coma disappeared suddenly, but the patient was delirious for several weeks and did not recognize anyone or his surroundings. This Marie regarded as like hysteria. The speech was slow and hesitating, and the visual fields were contracted.

---

[10] Parinaud. Annales d'Oculistique, Vol. 107, p. 283, 1892.

[11] Revue Neurologique, 1901, p. 428.

Parinaud pointed out that the downward movement in following a finger is a reflex act and better performed than a voluntary movement, and this he says he has observed in hysteria; but when the condition is hysterical there is no inconvenience from the ocular disturbance, just as there is no inconvenience from the contraction of the visual fields in hysteria, and Parinaud has never seen a hysterical patient incline the head forward in order to make use of the superior part of the *champ du regard* (Dr. de Schweinitz suggests as a suitable translation for this term, field of fixation). The dissociation between the voluntary and reflex movements is not always hysterical. Parinaud has pointed out that spasm may occur in sound ocular muscles as the result of paralysis of other ocular muscles when the patient is required to look in different directions. A paralysis confined to one eye may be the cause of spasm in the other, so that the sound muscles may appear to be affected. In these cases the paralysis is peripheral, but even in associated palsies it is not rare to see disturbance of the antagonistic muscles. In conjugate lateral paralysis from lesion of one sixth nucleus there is almost always nystagmus when the patient looks toward the sound side, and this nystagmus may be so pronounced that it may be difficult to say which side is affected. This I also have noticed in a case of complete paralysis of lateral associated movements on one side and of partial paralysis on the other. In the case which called forth so much discussion in the Neurological Society of Paris the visual fields were contracted only in the inferior portion and not concentrically, therefore the contraction was not like that of hysteria. The *champ visual* (visual field) Parinaud distinguishes from the *champ du regard* (field of fixation). He believes that if the cortical center for associated movements of the eyes in the frontal lobe be destroyed, the voluntary associated movements will be lost, but the reflex associated movements will be preserved, and yet little, if any, clinical evidence in support of this opinion can be found.

Babinski pointed out that in the case under discussion the downward movement of the eyes was imperfect with the head thrown back. He quoted the case reported by Schröder[12] in which only paralysis of the associated downward movement persisted, and the eyes could not be lowered below the horizontal plane. Any attempt to look downward caused a spasm of the elevators and eyes turned upward.

Oppenheim[13] has observed cases of pseudobulbar paralysis in which lateral movements of the eyeballs were impaired in voluntary innervation, but were preserved when the patient tried to follow an object or to turn toward the direction from which a sound came.

All the pathological evidence that I have been able to obtain in cases of persistent palsy of associated upward or downward movement is indicative of a lesion near the aqueduct of Sylvius. It is extremely doubtful whether a lesion confined to the corpora quadrigemina and causing no pressure on the surrounding parts ever causes paralysis of associated ocular movements, and those who favor such a view have not produced proof of a supranuclear center in this part. Bernheimer[14] says Topolanski showed that electrical irritation of the corpora quadrigemina of rabbits does not produce ocular movements, and that these parts could be removed without producing symptoms. He believed that there is a center for ocular movements near the oculomotor nucleus.

Bernheimer, from experiments on monkeys, has shown that the ocular movements do not depend on the integrity of the corpora quadrigemina, that this part of the brain does not contain a reflex center for these movements, and that after destruction of this part the ocular movements are normal. He obtained synergic ocular movements from irritation of the angular gyrus both before and after he had removed the corpora quadrigemina. He thus refutes Prus' statements that a center for ocular movements exists in the corpora quadrigemina.

---

[12] Schröder, quoted by Teillais, Bul. de la Soc. Fran. d'Ophthal., 1899, p. 415. (See my abstract of this case.)

[13] Oppenheim. Neurologisches Centralblatt, 1895, p. 40.

[14] Bernheimer. Wiener klin. Wochenschrift, No. 52, 1899, p. 1310.

Cases have been reported in which the corpora quadrigemina have been destroyed without disturbance in the movements of the eyeballs (Weinland, Seidel, Ruel, Nissen, cited by v. Kornilow).

In Case 2 of my series there was complete paralysis of right associated lateral movements, and great impairment of left and of upward associated movements. The paralysis of lateral associated movements is to be explained by the involvement of both posterior longitudinal bundles. If we assume that the nuclei of the superior rectus and inferior oblique muscles are in the posterior part of the oculo-motor nucleus, we can understand why marked impairment of upward movement was present in this case, but this is contrary to the views held by some concerning the position of these groups of nerve cells.

Siemerling[15] has recently remarked that the numerous experimental studies of Bach, Bernheimer, van Gehuchten and von Biervliet have not led to uniform conclusions. The oculomotor nucleus is merely the place for the transference of impulses and we are not in a position to state which parts are concerned with the innervation of the individual muscles.

My Case 1 is further evidence that the nuclei of the superior rectus and inferior oblique muscles may be in the posterior part of the oculomotor nucleus, because there was paresis of upward associated movement and the nerve cells of the oculomotor nuclei were not diseased, but the posterior part of the nuclei was affected by pressure from the tumor in the pons, and the aqueduct of Sylvius was compressed upon the left side. The tumor did not invade the nuclei of the third and fourth nerves. The paralysis of left associated lateral movement is explained by the almost complete destruction of the left half of the pons by a tubercle, and thereby involvement of the posterior longitudinal bundle. The left sixth nerve was also much degenerated. In both Cases 1 and 2 downward movement was normal, this would seem to be evidence that the nucleus of the inferior rectus muscle must be in advance of those for the superior rectus and inferior oblique muscles, as in each case the tumor grew from the pons and did not invade the oculomotor nucleus.

---

[15] Siemerling. Archiv. für Psychiatrie, Vol. 40, No. 1, p. 61.

If this is the correct position of these nuclei, a lesion developing ventrally in the pons as shown in two of my cases may by pressure upon the posterior part of the oculomotor nuclei cause paralysis of associated upward movement, as a result of injury of the nuclei for the superior rectus and inferior oblique muscles, and it would not be at all necessary to assume the existence of a coordinating center for upward associated movement. Paralysis of downward associated movement depends on impairment of the inferior rectus and superior oblique muscles, these having cells of origin in two distinct nuclei. There must be a close association between the nucleus of the superior oblique and that of the inferior rectus muscle of the same side, and possibly of the opposite side. It is probable therefore that a lesion of the nuclei of the inferior rectus muscles and of the fibers connecting them with the nuclei of the trochlear nerves would cause paralysis of associated downward movement, and it seems unnecessary to assume that a coordinating supranuclear center for upward or downward movements exist in the corpora quadrigemnia or their vicinity. In deed two of my cases (Cases 1 and 2) show that such a center is improbable, because in each case the lesion causing paresis of upward associated movement was posterior to the oculomotor nucleus, and did not implicate the corpora quadrigemina. Three of the cases of my series afford further evidence against a center for coordinate ocular movements in the corpora quadrigemina. In one the paralysis at first (Case 4) was only in upward movements, later also in lateral associated and in downward associated ocular movements. In this case therefore a paralysis at first confined to one form of associated movement later extended to other forms. In another case (Case 5) the paralysis of upward associated ocular movements disappeared, leaving only paresis of the left superior rectus, and finally this also disappeared. In Case 9 the associated palsy disappeared, leaving paralysis of muscles only on one side. It is very common to have a palsy of one or more branches of the oculo motor nerve in association with paralysis of associated upward or downward movement, and this is more easily explained by a lesion of the oculomotor nucleus than by a cortical lesion. Cortical centers control associated movements, not isolated muscles.

The paralysis of the sixth nerve occurring with paralysis of associated upward or downward movements has been attributed by Lichtheim to pressure upon the nerve at the base of the brain. The opinion held by Parinaud, although supported by Duval, Laborde and Graux, can hardly be accepted, viz., that the sixth nucleus gives nerve fibers to the internal rectus muscle, and that this muscle has a double innervation, the sixth nerve for lateral movement, the third for convergence.

The assumption of a basal coordinating center above the oculomotor nucleus for ocular movements seems forced. The lateral associated ocular movements can be well explained by the connection formed by the posterior longitudinal bundle between the nucleus of the branch to the internal rectus muscle and the nucleus of the sixth nerve. It is reasonable to suppose that certain fibers connecting the different parts of the oculomotor nucleus with one another, or connecting the group of nerve cells innervating the inferior rectus with the nucleus of the trochlear nerve have a coordinating function similar to that of the posterior longitudinal bundle, and that this function may be disturbed by a small lesion. It is striking that the paralysis of downward associated movement (necessitating the implication of two separate nuclei of ocular muscles) without paralysis of upward associated movement is exceedingly rare, far more so than is the isolated paralysis of upward associated movement.

Conjugate deviation of the eyeballs from lesions of the pons is not common when the lesion is confined to one side of the pons. In a case of hemorrhage into the tegmentum of the pons (see Case 3) that I have had the opportunity to study and the brain from which I have examined and refer to in this paper, inward deviation of only the right eye occurred from paralysis of the sixth nerve. One gets the impression that conjugate deviation of the eyeballs away from the side of the lesion is common in lesions of one side of the pons, but in four cases of lesions of this part observed by me (three of tumor and one of hemorrhage) causing paralysis of the external rectus muscle of the same side, I have looked for the conjugate deviation but failed to find it. It is probable that in most cases the side of the pons opposite to the lesion is directly or indirectly involved.

Von Monakow[16] believes that when the lesion implicates the sixth nucleus of one side, apart from the paralysis no disturbance, i.e., no deviation occurs, unless it is the result of secondary contracture. I believe that conjugate deviation is rare even as a late sign of paralysis of one sixth nucleus. Of course any involvement of both sixth nuclei would make conjugate deviation impossible, and in one of my cases (Case 2) both these nuclei were affected.

In a case of softening of the dorsal part of the left side of the pons reported by Albert Ransohoff[17] there was complete paralysis of the associated ocular movement to the left and of convergence movement of the right eye. The right external rectus muscle was not over-active, but the left internal muscle was in marked spasm. The left abducens nucleus was involved in the lesion, the right escaped, but the right posterior longitudinal bundle was involved. Ransohoff mentions that Wernicke and Jolly have shown that paralysis of the external rectus muscle does not in itself cause contraction of its antagonist, the internal rectus, and that in the cases of these authors, as well as in his own, there was a lesion of the opposite posterior longitudinal bundle. In Wernicke's case a tumor had destroyed the left abducens nucleus and invaded the right side of the pons. Both eyeballs were persistently turned to the right, especially the left eyeball.

According to the study of the literature by Ransohoff, contracture of the unparalyzed antagonistic muscles, but not always in both eyes, and more in the internal rectus muscle on the side of the lesion than in the external rectus muscle on the side opposite to the lesion, was observed in the cases of Wernicke, Jolly, Graux, Meyer (the lesion did not extend to the mid-line),

---

[16] Gehirnpathologique, p. 355.

[17] Ransohoff, Archiv. für Psychiatrie, Vol. 35, 1901, p. 416.

Spitzer (slight, and after one-half year duration of the lesion), Mierzejewsky and Rosenbach, Gebhard, and Ransohoff.

Ransohoff says that if the posterior longitudinal bundle, as is assumed by most authors, contains a part of the voluntary fibers for the internal rectus muscle of the opposite side, a lesion of these fibers, as of the pyramidal fibers, must cause spastic contracture of the muscle they innervate. Therefore, according to Ransohoff, contracture of the internal rectus muscle on the side of the lesion indicates that the posterior longitudinal bundle of the opposite side is also involved, but this contracture probably could not exist if the nucleus of each abducens were destroyed.

In four of my cases (Cases 1, 2, 3, and Beier) both posterior longitudinal bundles must have been much affected and conjugate deviation did not occur in any.

In one case (Case 4) in which paralysis of lateral movements was present, the movement of either eyeball to the right or left was distinctly greater when one eye was covered than in associated lateral movments. The individual movements of the eyeballs were therefore greater than the associated movements.

One of the cases (Case 9) was especially interesting in that syphilitic infection was positive. The paralysis of associated movement was peculiar; the downward movement was completely lost in the right eye and much impaired in the left; the upward movement was slight in the right eye and completely lost in the left; the lateral associated movements were nearly normal. Almost all the ocular movements were regained except the downward movement of the right eye. It is remarkable that the complete paralysis in the associated movements was in the downward direction in one eye and in the upward direction in the other, although the associated movement was very feeble. It is remarkable that the associated palsy disappeared leaving paralysis of downward movement of the right eye.

In four cases of my series with necropsy (Beier, Cases 1, 2, and 3) the pupil was smaller on the same side as the lesion of the pons. This alteration of the size of the pupil from a lesion of the pons is difficult to explain, but the presence of the sign in four cases shows that it is of diagnostic significance, and also that the fibers controlling the iris pass through the pons.

In at least two cases nystagmus was present in looking away from the side of the chief lesion (Beier, Case 2). This was caused by weakness of the opposite external rectus muscle from pressure upon or direct involvement of the nucleus of the abducent nerve. This sign may be expected when a lesion affects the dorsal part of the pons and destroys the function of one abducent nucleus and weakens that of the other. Nystagmus may surely be the result of paresis of ocular muscles.

I have studied 38 cases of paralysis of upward or downward associated movement reported in the literature, not including my own 9. In many cases certain symptoms are not referred to, so that statements regarding the frequency of symptoms are incomplete. V. Kornilow used only 20 cases of the 27 to which he found references in drawing his conclusions. As a result of my study of these 47 cases.

Paralysis of upward associated movement without paralysis or paresis of downward associated movement was found in 26 cases (Henoch, Priestley Smith (2), Nieden, Gowers, Parinaud (2), Ormerod, Reinhold, Thomsen, Lichtheim (3), Hoesslin, Bruner, Mills, Nogués and Sirol, Kornilow, Posey, Parkinson, cases 1, 2, 4, 5, 6, 8, of my series).

Paralysis of upward associated movement with paralysis or paresis of downward associated movement was found in 16 cases (Wernicke, Parinaud, Hope, Nothnagel, Eisenlohr, Verrey, Sharkey, Sauvineau, Teillais, Gordinier, Raymond and Cestan (slight), Kornilow, Cases 3, 4, 7, 9, of my series).

Paralysis of upward associated movement with impairment of lateral movement, often developing later, was found in 15 cases (Henoch, Priestley Smith (2), Lichtheim (3), Parinaud, Poulard, Raymond and Cestan, Kornilow, Parkinson, Cases 1, 2, 3 and 4 of my series).

Paralysis of upward associated movement without impairment of lateral movement was found in 22 cases (Wernicke, Nieden, Gowers, Parinaud (2), Ormerod, Hope, Thomsen, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Gordinier, Nogués and Sirol, Kornilow, Posey, Cases 5, 6, 7, 8, 9 of my series).

Paralysis of downward associated movement without paralysis of upward associated movement was found in 5 cases

(Gee, Poulard, Curzon-Marie and Babinski, Basevi, Schroeder), but in none of my nine cases.

The reaction of the iris was found to be impaired in 14 cases (Henoch, Gowers, Parinaud, Thomsen, Eisenlohr, Lichtheim, Sharkey, Sauvineau, Hoesslin, Gordinier, Mills, Kornilow, Posey, Case 9 of my series) and is said to have been normal in 4 cases (Parinaud, Teillais, Poulard, Raymond and Cestan), but in many cases no statements regarding the iritic reflex are made, and in all my nine cases the iritic reflex was preserved.

The optic nerve was found to be affected in 15 cases (Gowers, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Cases 1, 5, 7, 9 of my series), and normal in 12 cases (Wernicke, Nieden, Parinaud (2), Ormerod, Reinhold, Sauvineau, Teillais, Raymond and Cestan, Mills, Noguès and Sirol, Posey).

Convergence was impaired in 15 cases (Parinaud (2), Sharkey, Sauvineau, Teillais, Poulard, Gordinier, Raymond and Cestan, Kornilow, Basevi, Cases 2, 4, 5, 8, 9 of my series), and normal, or nearly normal, in 9 cases (Hope, Parinaud, Noguès and Sirol, Kornilow, Posey, Cases 1, 3, 6, 7 of my series.

Ptosis was found in 7 cases (Reinhold, Eisenlohr, Gordinier, Kornilow, Nothnagel, Cases 2 and 8 of my series), and was said to be absent in 13 cases (Wernicke, Parinaud, Gee, Sauvineau, Teillais, Raymond and Cestan, Cases 1, 3, 4, 5, 6, 7, 9 of my series).

Necropsy was obtained in 19 cases (Henoch, Wernicke, Gowers, Hope, Reinhold, Thomsen, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagle, Basevi, cases 1, 2 and 3 of my series.)

In all of these cases except one (Thomsen[18]) parts about the aqueduct of Sylvius were implicated, and in Thomsen's case a gumma was found in the cerebral peduncles. Thomsen cannot explain why the intense degeneration of the right oculomotor nerve and the slight degeneration of the left oculomotor nerve caused paralysis of only upward movement. It is possible that the Nissl method, if it had been employed, would have shown marked alteration of the oculomotor nuclei, possibly as a toxic effect from the adjacent gumma, and in this way the paralysis of upward associated movement may have been caused. Such was the condition in one of my cases (Case 2), unless perhaps the alteration of the nerve cells was caused by pressure. Until at least another case similar to Thomsen's is reported we may well hesitate to believe that a peripheral lesion of one oculomotor nerve may cause paralysis of only upward associated movements.

---

[18] Thomsen. Archiv. für Psychiatrie, Vol. XVIII., 1887, p. 616.

Tumor was found in 14 cases (Henoch, Gowers, Hope, Reinhold, Gee, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagel, cases 1 and 2 of my series), a bullet wound in one case (Eisenlohr), apoplectic cyst in one case (Wernicke), hemorrhage in one case (my Case 3), and lesion uncertain in one case (Basevi).

Recovery or partial recovery occurred in 7 cases (Priestley Smith, Nieden, Verrey, Kornilow, Cases 5, 8 and 9 of my series). Important symptoms other than ocular palsies were found in 41 cases (Henoch, Wernicke, Priestley Smith (2), Gowers, Parinaud (3), Hope, Reinhold, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Verrey, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Poulard, Gordinier, Cruzon-Marie and Babinski, Raymond and Cestan, Mills, Noguès and Sirol, Kornilow (2), Schroeder, Posey, Parkinson, Cases 1, 2, 3, 4, 6, 8 and 9 of my series).

As a result of my studies I believe that persisting paralysis of associated lateral movement indicates a lesion of the posterior longitudinal bundle; that persistent paralysis of associated upward or downward movement indicates a lesion in the vicinity of the oculomotor nucleus, and that paralysis of associated ocular movements is not the result of a lesion of extracerebral nerve fibers. Lesions of the cerebral cortex may certainly cause paralysis of lateral associated ocular movements, and possibly of upward or downward associated ocular movements, but cortical paralysis of associated ocular movements is transitory, unless possibly where the center on each side of the brain is destroyed. Paralysis of associated ocular movements may be caused by hysteria. Any case in which associated ocular palsy is persistent and is of organic nature, is unsuitable for operation unless the operation is merely palliative, as the lesion is probably within the posterior part of the pons or cerebral peduncle, according to the form of the associated palsy, or else causes much pressure upon the dorsal portions of these structures. The paralysis of associated ocular muscles may be produced by inflammatory lesions or lesions of a similar character (alcohol, syphilis) as well as by tumor, and may disappear later in the course of the disease. Syphilitic ependymitis and cellular infiltration must always be considered in diagnosticating the lesion causing paralysis of associated ocular movements.

CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT OBSERVED BY DR. SPILLER.

Case I. Tubercle of left side of the pons.

Galik, was admitted to my wards in the Philadelphia General Hospital, Aug. 11, 1904, and died Oct. 22, 1904.

He said he had been paralyzed for nine months on the right side of the body. He was a fairly well developed man, aged thirty-one years. He was examined Aug. 12 by Dr. T. H. Weisenburg, when the following notes were made:

He never had headache nor vertigo, and has had no difficulty in chewing or swallowing, this statement being corroborated by the nurse. There is no dribbling of saliva. He has internal deviation of both eyes, more of the left. The right pupil is larger than normal, the left is normal in size. Both pupils respond to light and in accommodation and convergence. The left external rectus is completely paralyzed, and the right external rectus is partially paralyzed. All of the other movements are well performed. On showing his teeth he can draw back the right angle of his mouth better than the left. The right nasolabial fold is more distinct than the left. On attempting to close his eyes the left lids remain partially separated. He wrinkles the right side of the forehead normally, but there is hardly any movement on the left side. The tongue deviates to the right when protruded, but is not atrophied and shows no tremor. The masseter muscle does not contract so well on the left side as on the right.

He is able to lift the right upper limb from the bed for a few minutes, but the movement is unsteady and weak. Resistance to passive movement at the right elbow is almost lost. The right upper limb seems somewhat spastic. The right forearm is held in supination, the hand flexed on the wrist, and the fingers flexed into the palm. The grip is very feeble. There are no contractures, but the fingers are stiff. The whole right upper limb is wasted, more so in the distal parts, the muscles of the thenar and hypothenar eminences being almost completely atrophied. Atrophy of the extensors and flexors of the forearm is more marked on the right side than it is in the corresponding muscles of the left upper limb. The biceps, triceps and wrist reflexes on the right side are prompter than normal.

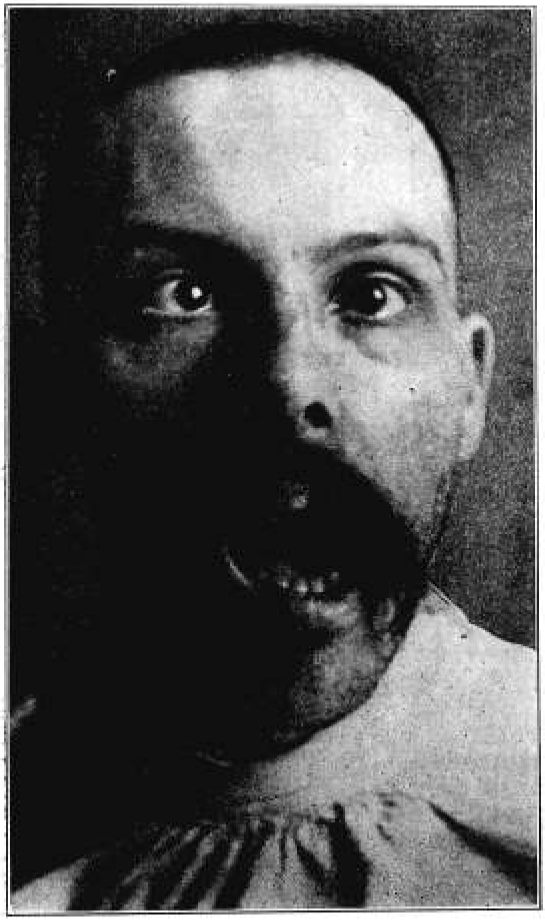

FIG. 1. Internal deviation of both eyes, especially of the left, and deviation of the lower jaw to the left from paralysis of the left muscles of mastication. Paresis of associated movements upward and to the left.

Sensation for touch is felt in the right upper limb, right side of chest and abdomen, but less so than in the left side. He does not recognize a pin prick over these parts on the right side as a pin prick, but as touch, and sensation is more impaired in the distal parts. The left upper limb is normal in every respect. He is not able to recognize objects placed in his right hand.

The power of the right lower limb is diminished. He is able to flex and extend the thigh, but is not able to move the ankle joint or the toes. The motor power in the left lower limb is normal. The patellar and Achilles tendon reflexes are much increased on the right side, but not on the left. Sensations for touch and pain are diminished in the right lower limb, and more so in the distal parts; they are normal in the left lower limb. The right lower limb is somewhat spastic and atrophied, and the atrophy is seen especially in the thigh muscles. He has had no bladder or rectal disturbances.

He says he has no pain anywhere.

His speech is thick and indistinct.

Dr. Van Epps saw the patient Aug. 13, and made the following notes: Contraction of the left orbicularis palpebrarum is markedly weakened, that of right is questionably weakened. Right pupil is round, 3 mm. in size. Left pupil is round, 2 mm. in size. Both pupils react to light and in accommodation and convergence. Outward rotation of O. D. 2 mm. short; internal rotation 3 mm. short, and upward 2 mm. short of full; downward rotation normal.

O. S. inward rotation normal, complete absence of outward rotation, upward and downward rotation similar to that of fellow. On this date some weakness of the right orbicularis palpebrarum was noticed. The nurse said the patient had difficulty in swallowing.

Dr. Van Epps made a further report Aug. 16, 1904.

O. D. Under homatropine, pupil 6 mm. round; media clear, marked optic neuritis with swelling of retina for 1½, disk clean in all directions (neuroretinitis). Disk elevation 2 diopters. Arteries reduced in size, veins slightly increased in caliber and quite tortuous, partly disappearing here and there in swollen nerve and retina. Hemorrhage on upper part of disk. Middle and peripheral zones normal. O. S. Under homatropine 4½ mm., round. Cornea epithelium so eroded that a view of the fundus is impossible.

Entire loss of corneal and conjunctional reflex on the left side (hemianesthesia of same complete).

There seems to be marked hyperesthesia of eyelids and surrounding cutaneous area on the left side, but patient's answers are contradictory.

He was seen by me Aug. 25, at which time I made the following notes:

In converging the movement of the right eyeball inward is about equal to that of the left, and is greater than when the attempt is made to look toward the left with both eyeballs. He has paralysis of associated movements, both in looking to the left and upward. The movement of each eyeball upward is impaired equally. When he closes his eyelids he has marked fibrillary tremor of the left upper lid. He has marked fibrillary tremors of the left masseter and the left masseter is completely paralyzed. When the mouth is opened the jaw goes markedly toward the left. Speech is distinctly bulbar. Hearing cannot be tested carefully on account of slow cerebration, but seems to be affected on the left side. The weakness of the right upper and lower limbs is very pronounced, though it is not complete paralysis. The tendon reflexes of the right upper and lower limbs are distinctly exaggerated.

He is able to stand, but not without support.

He swallows with difficulty.

The left cornea is anesthetic.

He says pin prick is felt more in the left side of the face. He draws up the corner of the mouth when the corresponding side of the face is stuck with a pin. This sensory condition was noticed also by Dr. Weisenburg, who found (Aug. 12, 1904) that he recognized touch and pain sensations distinctly better on the left side of the face than on the right.

Sept. 21. Speech is very indistinct and is bulbar in character. The left side of the face is paralyzed. Marked fibrillary tremors are seen in both sides of the tongue, and the tongue deviates to the right when protruded. The left pupil does not reach the median line, and the right pupil goes a little beyond the median line in trying to look to the left. Pain and tactile sensations are diminished in the right upper limb. Babinski's sign is absent on each side. Ankle clonus is not present on either side.

Sept. 23. He does not hear a low ticking watch when placed on the left ear, but does hear it when it is placed on the right ear. He has excessive appetite.

Sept. 28. The left eye is much congested, although mosquito netting is kept constantly over his face to keep the flies away and his eyes are washed frequently with boric acid. Pus is found in the anterior chamber of the eye.

Oct. 22. He has been gradually sinking more than ten days. Respirations have become shallow and rapid, and he is unable to speak. He died at 8.30 p.m., Oct. 22, 1904.

Death occurred while the patient was in the service of Dr. C. K. Mills, after I had gone off duty. I am indebted to Dr. Mills for the pathological material.

At the necropsy ulcerative tuberculosis of the lungs and a large tumor in the left side of the pons were found.

Sections made through the cerebral peduncles show that the aqueduct of Sylvius is compressed on the left side. The left cerebral peduncle is considerably large than the right. The tumor extends into the lower part of the left cerebral peduncle. The left fourth nucleus appears to be almost normal, a few of the cells are slightly altered, but the nucleus on the whole is remarkably well preserved. The posterior longitudinal bundle on the left side is pushed more dorsally than is the corresponding bundle on the right side. The right 4th nucleus also is normal except that a few of the cells show alteration. Most of the nerve cells in the oculomotor nucleus, both in each lateral and in the median group seem to be normal, only occasionally a cell can be found in which the chromophilic elements have undergone disintegration.

Left 2nd nerve. There is a slight round cell infiltration of the pia and of the septa extending into the optic nerve; the nerve does not appear to be degenerated in its intracranial portion.

Left 3d nerve appears to be entirely normal.

Left 6th nerve is much degenerated. The medullary sheaths are swollen. The axis cylinders are also swollen and in some places have disappeared. One side of the nerve is much more altered than the other. The Weigert stain shows much degeneration at one side of the nerve where the medullary sheaths stain very badly. The right sixth nerve appears to be normal.

Left 7th nerve shows a moderate swelling of medullary sheaths.

Left 8th nerve appears to be normal.

Left 5th nerve is almost completely degenerated in a few bundles that probably belong to the motor part of the nerve.

The left pyramid is much degenerated by the method of Marchi, as is also the spinal root of the left fifth nerve. There are some degenerated fibers on each side of the medulla oblongata posterior to the lower olive, more on the left side, and also in the left lemniscus.

The right crossed pyramidal tract is much degenerated by the Marchi method from the cervical into the lumbar region. The left direct pyramidal tract is much degenerated in the cervical region.

There is no meningitis about the medulla oblongata or spinal cord. The nerve cells of the anterior horns of the cervical and lumbar regions stain well by the thionin method.

Weigert hematoxylin sections show intense degeneration of the left anterior pyramid, of the left direct pyramidal tract, and of the right crossed pyramidal tract.

The tumor in the middle portion of the pons, antero-posteriorly, invades a little the right side of the pons (see Fig. 2), but the greater part of the right pyramidal tract is not involved in the growth. The aqueduct of Sylvius is much compressed and appears as a long narrow slit. Almost the entire left half of the pons is destroyed, a narrow zone at the ventral part escaping. The tumor in transverse section is nearly round. Microscopically the tumor is not sharply defined from the surrounding tissue, and its peripheral zones consist of mononuclear cells with round nuclei. The central portion of the tumor is degenerated, and the cells do not stain well. The tubercle bacillus was not found. The tumor has the appearance of a tubercle.

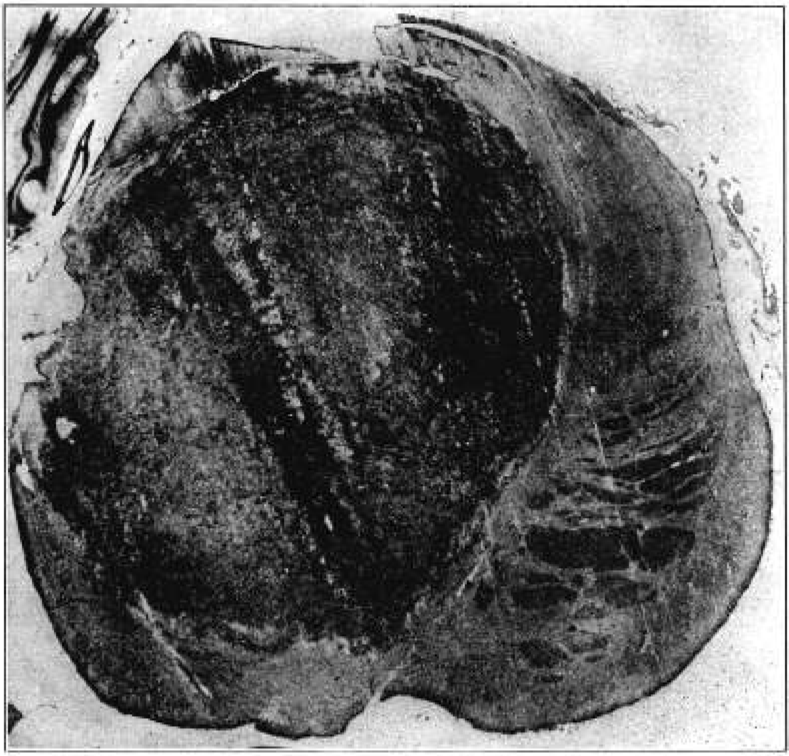

FIG. 2. Tubercle in the left side of the pons.

Summary: B. Galik, a male aged thirty-one years, had been paretic (Aug. 11, 1904) nine months in the right limbs. He was paretic in both sides of his face, especially in the left side, and in the left muscles of mastication, and had fibrillary tremors of the left masseter muscle, and of the right side of the tongue. The tendon reflexes in the right upper and lower limbs were exaggerated. Diminution of sensation and some wasting were observed in the right upper and lower limbs. The movements of both eyeballs upward were impaired, and there was complete loss of movement of the left eyeball to the left, and marked impairment of movement of right eyeball to the left; also, impairment of movement of right eyeball, but not of left eyeball, to the right. Downward movement of both eyeballs was normal, and convergence was preserved (paresis of associated movements upward and to the left). He had bulbar speech, impaired hearing in the left ear, anesthesia of the left cornea, optic neuritis, at least in the right eye (left disc not visible because of corneal opacities); and later left neuroparalytic keratitis.

The necropsy showed a large tubercle involving almost the entire left side of the pons and invading a little the right half of the pons and extending into the lower part of the left cerebral peduncle. Most of the nerve cells of the oculomotor nuclei appeared to be normal, but those of the left nucleus were displaced. The left 3d nerve was normal. The left sixth nerve and spinal root of the left 5th nerve were much degenerated, and the left 7th and left motor 5th nerves were partially degenerated. The left pyramid was degenerated. The aqueduct of Sylvius was much compressed from the left side.

The slight voluntary movements of the right upper and lower limbs may be explained by the preservation of a narrow zone in the ventral portion of the left half of the pons and by axis cylinders that probably passed through the tubercle without being destroyed. It has been shown that axis cylinders may be found within a tubercle even after the medullary sheaths have been destroyed.

The paralysis of associated left lateral movement is to be explained by the implication of the left posterior longitudinal bundle. The paresis of upward movement could not have been caused by implication of the corpora quadrigemina, because this portion of the brain stem was not involved. The nerve cells of the oculomotor nuclei were slightly degenerated, but the left oculomotor nucleus was much displaced in its posterior portion, and the function of the nerve cells here must have been interfered with.

The pupil in this case was smaller on the side of the tumor.

It seems difficult to explain the absence of paralysis of associated ocular movement to the right: unless it is because the ocular examination was made two months before death occurred, and at that time the turbercle had not extended to the right side of the pons. The man's intelligence became considerably affected before death.

Case 2. Glioma of the right side of the pons.

A. Moore, male, aged sixteen years, was a patient of Dr. Wm. Evans, from whom the following notes were obtained:

His parents and two sisters were living and well. His maternal grandfather died of apoplexy at seventy-one years. He had always had good health until the early part of July, 1904, when he began to feel nervous, slight exertion made him tired, he became dizzy, had headache and a tendency to turn to the left, and he staggered slightly in walking. His sight became dim, and at times he had diplopia.

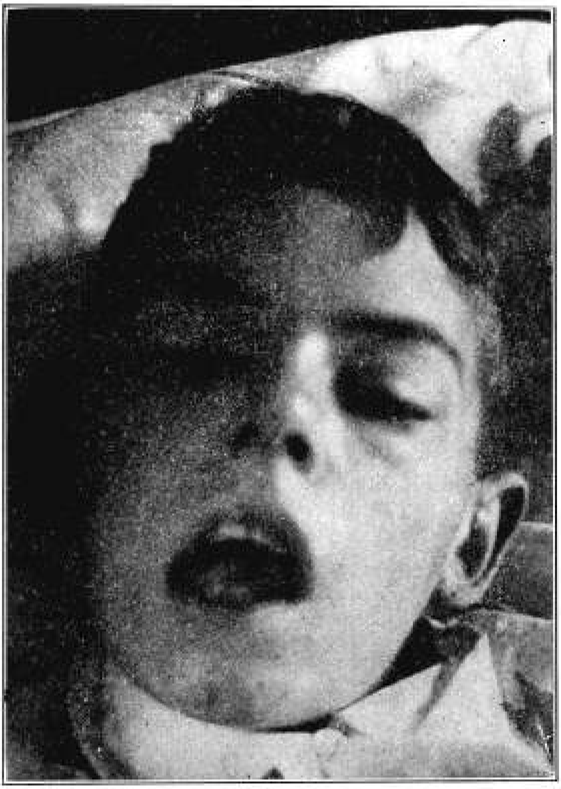

FIG. 3. Attempt to converge. Notice position of left eye. The left eye is moved more toward the right than when the attempt is made to move both eyeballs to the right (see Fig. 5). Notice the marked deviation of the lower jaw to the right when the mouth is opened, this being the result of paralysis of the right external pterygoid muscle.

He was seen by Dr. C. S. Potts Aug. 15, 1904, and notes made by him at that time are as follows:

He sits with the head retracted and bent to the left, the chin pointing to the right, although he can hold it straight if he makes an effort to do so. He has ptosis of the left eyelid. The pupils are equal and respond to light and in accommodation. Some weakness of the external rectus of the right side is detected and he says that he sees double when he looks to the right. Coarse nystagmic movements are present when the eyes are moved laterally in either direction. The angle of

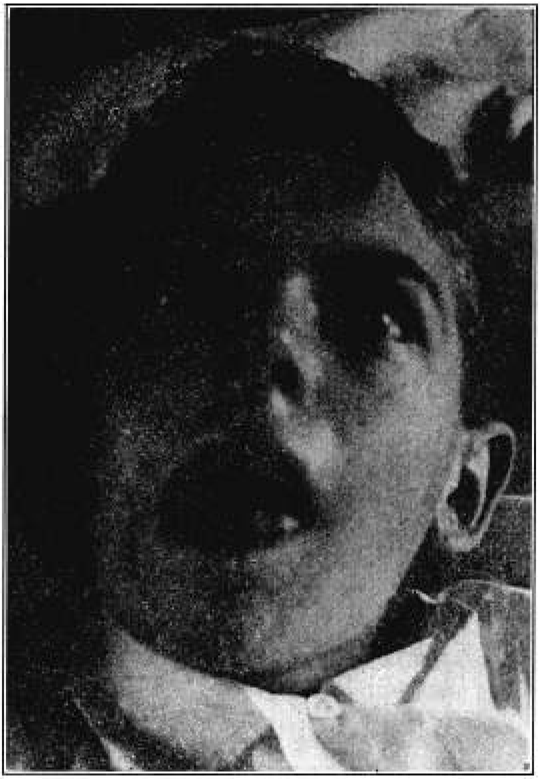

FIG. 4. Attempt to look upward and to protrude the tongue. The eyeballs move very slightly upward. The lower jaw goes to the right, the tongue to the left when protruded. The dot on tongue shows the point of the tongue. On great effort, the tongue could be protruded a little further, and the deviation to the left was greater.

the mouth droops slightly on the right side, but there is no distinct paralysis. The tongue seems to protrude slightly to the left. The gait is staggering, but there is no tendency to fall to one side more than the other. Resistance to passive movements is good. The patellar reflexes are exaggerated, the left more than the right, and he has slight ankle clonus on the right side and marked clonus on the left side. Plantar reflexes are diminished, the toes move in neither direction on the right side, but slightly upward on the left side. The arm jerks (biceps, triceps, wrist) are apparently equal on both sides and about normal in degree. He hears the tick of a watch in the right ear at 12 inches, in the left ear at 3 feet. Bone conduction is better on the left side. He can recognize the direction of passive movements of the fingers and toes, but cannot touch the end of his nose with either index finger with certainty. He has not other sensory symptoms except headache.

An examination of the eyes by Dr. Krall, Aug. 17, 1904, gave the following results:

He has partial ptosis of left eye, paralysis of the left external rectus, or rather a paresis, paresis of the left inferior rectus, and a nystagmus to the right on attempting fixation. Accommodation for his age is good. The pupils are nearly alike on the average (the left one is ½ mm. larger than the right), they react normally to light, accommodation and convergence. His vision is practically normal. The ophthalmoscopic examination shows no involvement of the optic nerve. Field of vision is, however, contracted concentrically for form and color. Color perception is good. No color blindness.

The patient was seen by me Oct. 11, 1904, in consultation with Dr. Evans, and at that time I made the following notes:

The left pupil is considerably larger than the right, Iritic reflex is prompt in each eye. Nystagmoid movement in trying to look to the left. Movement of eyeballs to the left imperfect. Left iris does not reach the external canthus and the eyeball returns almost immediately towards the median line. Unable to look towards the right with either eye, pupils remaining in the median line when he attempts to look to the right. Able to converge very slightly with the left eye, and does so with the right. Upward movement of each eye is almost lost, the pupils hardly passing beyond a line drawn horizontally across the centers of the palpebral fissures. Downward movement seems almost normal. He closes the eyelids firmly on each side. Slight drooping of each lid is present. He wrinkles the forehead well and equally on both sides. The movements of the face are feeble on each side in the lower distribution of the seventh nerve. He does not show the teeth well nor does he draw up either corner of the mouth well. Tongue when protruded goes distinctly towards the left. Left masseter muscle contracts, the right apparently does not. When the mouth is opened the lower jaw goes far to the right. The right orbicularis palpebrarum is distinctly weaker than the left, and often in closing his lids, the right lids remain partially open. Pin prick is promptly perceived on each side of the face. It is probable that the right side of the face in the lower distribution of the facial nerve is a little weaker than the left side. The neck is not stiff. There is a tendency for the head to turn to the left, but it can be turned easily to the right. He hears a low-ticking watch 6 to 8 inches in the left ear. Cannot hear it in the right ear when placed against the ear. Speech is thick, bulbar in type, not at all aphasic. He counts fingers correctly

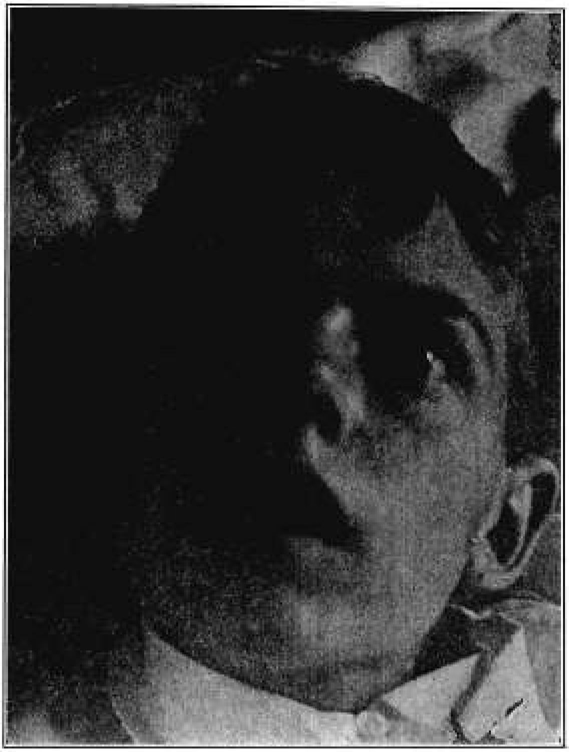

Fig. 5. Attempt to look to the right and to separate the lips with the teeth together, showing weakness in the lower distribution of the facial nerve on each side. Notice the position of the left eye in this photograph and in Fig. 3, showing attempted convergence.

held before either eye. He is able to swallow liquids or semisolids without much trouble. The soft palate moves freely on voluntary effort. Finger-to-nose test shows extreme ataxia with right hand. The left upper limb is almost completely paralyzed, but there is slight movement of the left shoulder. No motor power is preserved below the shoulder in the left upper limb. Biceps and triceps reflexes are slightly increased on the left side, almost normal on the right side. Sensations for touch and pain are somewhat diminished in the left upper limb, and are normal in the right upper limb. Sense of position seems to be preserved in the left upper limb, as he puts his right hand directly on his left with eyes closed, when told to do so. Upper

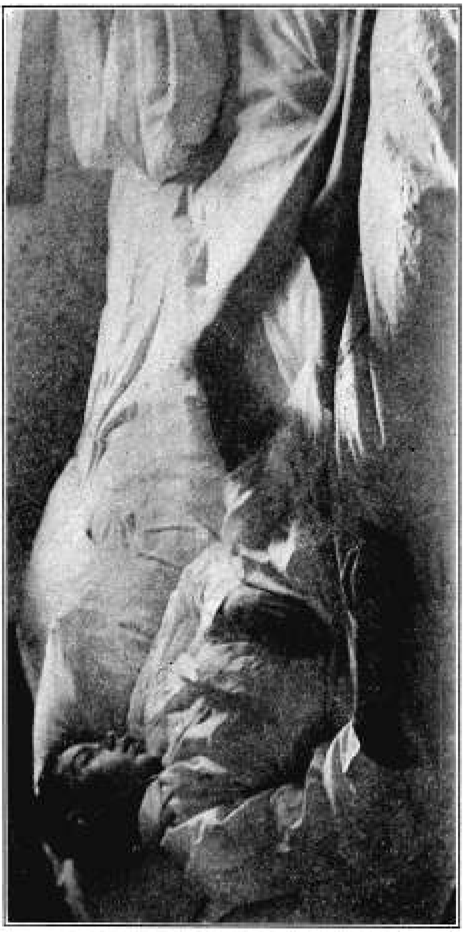

Fig. 6. Showing the tendency of the head to fall forward and to the left; also the complete paralysis of the left upper limb and the partial voluntary movement of the left lower limb at the hip. The voluntary power at the left shoulder was very slight.

limbs are not distinctly wasted. Left lower limb is distinctly weaker than the right, but not as weak as the left upper limb. The right lower limb has normal power. The lower limbs are not distinctly wasted although the whole body is somewhat emaciated. The patellar reflex is slightly increased on the right side, distinctly so on the left. Tendency to patellar clonus is present on the left side only. Persistent ankle clonus on both sides, but much more distinct on the left side. Typical Babinski sign is obtained on the left side, but not on the right side, there being no movement of the toes at all on this side. Sensations for touch and pain are probably diminished slightly in left lower limb. Heel to knee test shows much ataxia of right lower limb. When sitting on the edge of the bed he would fall if not supported, and head falls far forward, and to the left. When made to stand he would fall if not supported. He cannot stand erect, and ataxia is so great that he does not seem to know where to put his legs. His knees flex under him. He understands all that is said to him. Sitting up makes him dizzy. He has ocasional attacks of dull headache in the right temporal region. The tongue is not wasted, nor does it show fibrillary tremor. Almost every day he has attacks of nausea, sometimes with vomiting.

In converging left eyeball moves distinctly a little more toward the right than when he tries to look to the right with both eyeballs.

Further notes were made by me Oct. 23, 1904.

Upward movements of eyeballs much impaired, but seem to be better than when he was last seen. The jaw goes far to the right. Tongue not protruded much beyond the lips, but deviation to the left occurs. Movement of eyeballs to the right is lost, much impaired toward left and nystagmus occurs when eyes are turned toward the left. Weakness of right orbicularis palpebrarum is more pronounced. Right eyeball moves up a little more than does the left. He raises the left eyebrow better than the right. He does not hear a loud-ticking watch unless placed directly on the right ear, but hears it a foot away from the left ear. Sensations for touch and pain are not much impaired on either side of the face. He certainly feels touch and pain in the left forearm. The movement in the left lower limb is almost entirely at the hip. He feels both touch and pain sensation in the left lower limb. He has no movement in the left toes or at the left ankle. When he sits up the head falls far forward and to the left and the whole body inclines forward.

The necropsy was obtained Nov. 10, 1904.

The left 4th nucleus is partly degenerated, some of the nerve cells of this nucleus show much degeneration, but most of them are in excellent condition. There is no distinct group of cells constituting the right 4th nucleus at this level, because the right 4th nucleus has been pushed more cerebralwards by the tumor. The right posterior longitudinal bundle has been pushed dorsally toward the aqueduct of Sylvius, and the aqueduct is distorted in such a way that its right half is pushed further dorsally than its left half. The glioma extends into the right cerebral peduncle as high as the level of the fourth nucleus, but does not invade this nucleus. Some of the cells of the right fourth nucleus are degenerated, but most of them are normal.

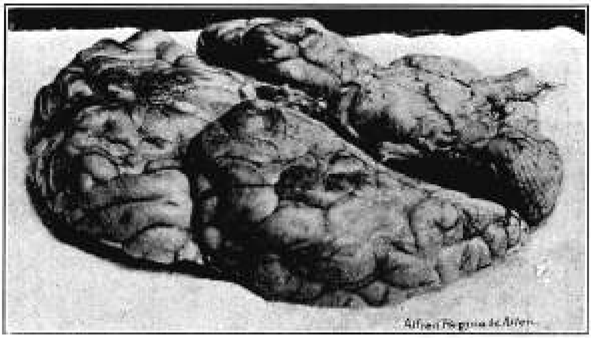

FIG. 7. Glioma of the right half of the pons, invading the lower left half of the pons.

The median group of cells in the oculomotor nucleus is intensely altered, the chromophilic elements have become fine granules and the nuclei are displaced far to the periphery of the nerve cells. The left lateral group in the oculomotor nucleus is well developed, although here and there a nerve cell is found that is not quite normal. The right lateral group shows much alteration, and yet not nearly so much as the median group. The nerve cells are much less numerous in the right lateral group than in the left lateral group, and they are pushed backward toward the aqueduct of Sylvius; many of them appear to be normal. Cells bordering on the upper and median side of the right lateral group are intensely degenerated. The right oculomotor nerve in its extrapeduncular portion does not appear to be degenerated.

The right second, fifth and sixth nerves and the left sixth nerve do not appear to be degenerated either by the Weigert hematoxylin or acid fuchsine method.

The right half of the medulla oblongata is much larger than the left, but does not contain any tumor tissue at the level of the twelfth nuclei. The twelfth nuclei and the intramedullary part of the twelfth nerves appear to be normal. The right anterior pyramid takes the Weigert hematoxylin stain a little less deeply than the left, but the degeneration is slight.

Sections from the medulla oblongata stained by the Marchi method show some recent degeneration of the right anterior pyramid, and of each column of Burdach, but not of the columns of Goll.

Sections from the uppermost part of the cervical cord show considerable recent degeneration of the left crossed pyramidal tract, and of the columns of Burdach, and slight recent degeneration of the right crossed pyramidal tract.

The comparatively slight degeneration of the pyramidal tract from the right side of the brain is striking in consideration of the size of the tumor.

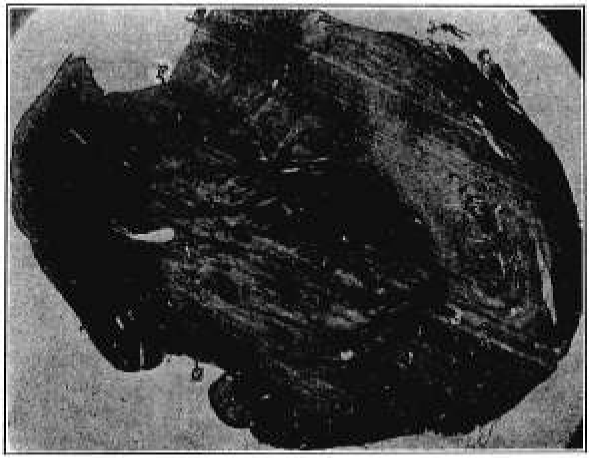

FIG. 8. Microscopical section through the upper part of the pons, showing the glioma of the right half of the pons. FG=raphe of pons.

A section made through the upper part of the pons shows that the tumor is entirely in the right half of the pons, does not reach the raphe, and leaves intact many bundles of the pyramidal tract on the right side, although they are more widely separated from one another than is normal. The right lemniscus is not degenerated. The tumor is chiefly in the lateral part of the right side of the pons, and here is cystic. The tumor consists of cells containing small round or slightly oval nuclei and are separated from one another by much intercellular fibrous tissue. The tumor is a glioma, and in some places is very vascular. It invades the left side of the pons nearer the medulla oblongata.

Summary: A. Moore, male, aged fifteen years, (Oct. 11, 1904) had been complaining about nine weeks. The left pupil was larger than the right. Iritic reflexes prompt. The movements of the eyeballs to the left were imperfect, and to the right lost. Upward movement was much impaired, downward movement about normal. Convergence preserved, but impaired. (Paralysis of upward and lateral associated movements.) Slight bilateral ptosis. Weakness of each facial nerve and of the left side of the tongue. Paralysis of the right masseter muscle and of the left limbs. Tendency to fall to the left. Impairment of hearing in the right ear. Bulbar speech. Diminution of sensation in the left limbs.

A large glioma was found in the right half of the pons, extending into the right cerebral peduncle and the right side of the medulla oblongata, and in the lower part of the pons involving the left side of the tegmentum of the pons. The nerve cells of the 4th and 3d nuclei were partly degenerated, especially on the right side. The left crossed pyramidal tract showed considerable recent degeneration.

The slight impairment of sensation may be explained by the slight involvement of the lemniscus, but it is remarkable that the left motor paralysis was so intense when so much of the motor tract in the right side of the pons escaped. In the left upper limb the only voluntary movement was slight drawing up of the shoulder, and in the left lower limb slight flexion at hip and knee.

(To be continued)